# THE DURATION OF LIFE IN CASES OF INFECTIVE ENDOCARDITIS.

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Two examples of this disease, in which I have recently had opportunities of verifying the diagnosis at the autopsies, throw some light on the duration of life in these cases. Their histories are briefly as follow. See also Appendix to previous paper, p. 377.

History of Case, No. 61.—The patient, a single woman, had had feeble health for 5 years past, at which date she had an attack of "rheumatic fever," and was an in-patient of the Sussex County Hospital. She had been subsequently readmitted several times as an in-patient, suffering from heart troubles, pains in joints, epistaxis, vomiting, and so forth. On one occasion she had paresis of some of the muscles of the left fore-arm and hand. When admitted for the last time in September 1894, the heart's apex was found displaced outwards to nipple vertical in fifth interspace; the area of dulness was somewhat increased, and a loud double murmur was heard over the aortic cartilage, and less loudly towards the apex; the bases of lungs were subcrepitant; both spleen and liver were palpable below the margin of ribs; the legs were swollen at times, and the urine, sp. gr. 10·10, contained albumen. The average daily amount passed towards the end of life was 20 oz.; she was then troubled with persistent vomiting. The temperature was variable; on most days it was 3 or 4 degrees above normal for a few hours. She died of exhaustion, 25th October.

History of Case, No. 62.—About 2 years before his death the patient had an attack of "rheumatic gout," and some weeks subsequently he came under observation as an in-patient with left hemiplegia and heart disease. At the cardiac apex, which was displaced outwards, there was a presystolic murmur. After a 3 months' stay in the hospital, he, to a large extent, recovered the use of his limbs, and was discharged; but he returned again at the end of 5 weeks with paresis of the left arm and hand. This second attack came on suddenly after a "bolster match" with his brother. The heart's apex was 2 in. outside nipple line in fifth space. The presystolic murmur remained; temperature about normal; there was no albumen in the urine. After a month's stay in hospital he was discharged with almost complete recovery of the use of the paralysed muscles. He was finally admitted about a year afterwards, with orthopnœa, œdema of legs, and persistent vomiting. The heart apex beat was in the sixth space, considerably outside the nipple line; its action was hurried and irregular; the pulse, 96, was of low tension; temperature 100° F.; the urine was very albuminous. He died 4 days after the last admission.

One patient, a girl, first came under observation when she was 17 years of age, and was repeatedly seen as an in-patient by me until the time of her death 5 years afterwards. In all she had four severe attacks of illness during that period. Of these the earlier attacks were diagnosed "acute rheumatism," as they usually are when the cardiac symptoms are accompanied with some arthritis. On carefully reviewing the clinical histories of her various attacks I am, however, now confirmed in the opinion that throughout her illness I was watching and treating progressive outbursts of identical pathological processes (using the term in the sensc recently adopted by Dr. Semon), for the following reasons. She had heart trouble from the commencement; she had repeatedly, throughout her attacks, pain, redness, and swelling of the knees, shoulders, and hands. At an early date she became subject to epistaxis, and bleeding from the gums. On her penultimate visit to the hospital as an in-patient, 4 months before her death, she had loss of power in the left forearm and hand, due probably to an embolus. And, finally, in her last attack, her symptoms were ascribable to the failure of a long wearied and a much worried heart to carry on the work of the circulation. After death, besides considerable hypertrophy of the left ventricle, there was upon the auricular surface of the aortic cusp of the mitral valve a tough fleshy growth about half an inch long, and an old superficial ulceration of the wall of the left auricle. The aortic cusps were shortened and thickened. There was an old infarct in the right kidney.

The second case, that of a young man, an undertaker by trade, is in many respects similar to the above. The duration of life after the history of an attack of "rheumatic gout" was, however, shorter, certainly not more than two years, if so long. He was an in-patient on three separate occasions. Here again there was progressive heart disease from the first, with the early appearance of unilateral paralysis, which a prolonged stay in the hospital "cured." He soon, however, returned with renewed signs of arterial plugging, to be again discharged cured. In this example of infective endocarditis, unlike the other, the early stages of the disease were not associated with high temperatures and severe arthritic symptoms. There were, however, occasional attacks of pain and swelling of the right ankle, the "rheumatic gout," but there was no history of mucous hæmorrhages recorded in the notes. The final symptoms, the breakdown in the cardiac muscle, the persistent vomiting, the anasarca, the albuminous urine, and the congested lungs were closely alike in both instances. The autopsy showed from the size and weight (23 oz.) of the heart of the youth, that the disease of the valves was a chronic one, a conclusion confirmed by the clinical history. It also showed the heart at the time of death to have been pathologically in a quiescent state; that is to say, the firmly coherent mitral flaps with their contracted orifice, fringed by short fleshy growths, were the passive results of an

acute process, which had then ceased. Infective endocarditis consists, I take it, essentially of a series of sudden outbursts of acute disease, followed by prolonged intervals of healthy calm, during which a youthful patient can apparently repair more or less fully any structural flaws, induced by the acute attacks.[1] If this view of the disease is admitted to be the true one, and I cannot find any other interpretation so applicable to the various clinical and pathological facts of cases under my care, the resemblance between infective endocarditis and acute rheumatism, in these as in many other respects, is close and striking. Infective endocarditis is, however, admittedly a disease associated with the presence of microbes within the vascular system, whilst the relationship between these bodies and acute rheumatism is at all events doubtful. As fatal cases of acute rheumatism without heart mischief must be exceedingly rare, and will include inflammations of the lungs and pleural cavities when pneumococci usually abound, the etiological uncertainty of this disease is readily explained.

The student upon his first visit to a bacteriological laboratory cannot fail to note how difficult it is to obtain a pure culture of a bacillary growth. What untiring vigilance has to be exercised, what elaborate precautions have to be taken by the manipulator to prevent the introduction into the nutrient jelly of other germs than those he seeks to cultivate. Much of the difficulty, I admit, has been overcome by routine procedure, yet this only emphasises the fact that nature in her profusion sows the face of the jelly with air-borne germs of many species, unless she is prevented by man. The valvular growths found within the heart in infective endocarditis are admittedly the home of many different species of bacteria in different subjects.[2] Not only is this the case, but several species of cocci may occur in the same subject And this bacterial commixture of species is not confined to the endocardial growths, it is common to other diseases, to the teeming membranes of diphtheria, and the rice-water stools of cholera;[3] as we might expect from the experience of the laboratory. There is, however, this important distinction between these examples of mixed infection. In the two last the culture beds of the microbes within the body may be, and probably often are, directly contaminated through the air; whilst the first, by the position of the heart and the great

[1] Drs. Pye-Smith and Frederick Taylor, amongst other writers, have recorded cases of so-called recovery from infective endocarditis. The most curious part about these recorded cases is their rarity, and for this result the nomenclature of the early stages of the disease is largely responsible.

[2] Tubercle bacilli, the *Diplococcus pneumoniæ*, the anthrax bacillus (Oliver), Typhus bacilli (Dreyfus-Brisac), besides the streptococci and staphylococci of pus, have been found by different observers within the heart or aorta in this disease. Fränkel and Sänger found five different pathogenic microbes in thirteen cases of endocarditis (ulcerosa and verrucosa). Kanthack says there are at least twelve different organisms met with in ulcerative endocarditis!

[3] Diphtheria (Klebs-Loeffler) bacilli are frequently associated with pus cocci, *Spirillum choleræ asiaticæ* with Finkler-Prior spirillum (Weichselbaum).

vessels, removes its culture beds as far as possible from all sources of direct contamination; yet we know that even here within the heart, whither germs can only be conveyed by the hæmolymph system, mixed infection so frequently occurs as to be the rule rather than the exception. It would seem probable that as soon as a swarm of pathogenic microbes have succeeded in forcing a passage through the body's chief lines of defence against such attacks, namely, the skin and the mucous membrane, others of the same or of different species can readily follow.

If the distinguishing peculiarities of each mycotic disease is due to some specific differences in the pathogenic microbe,—its etiological factor,—as some bacteriologists assume, the wonder is, when we consider the ease with which a mixed infection can take place that these specific disorders have any constant characters of their own, not that the characters occasionally vary. These remarks apply especially to cases of infective endocarditis, where there is apparently no specific microbe, and where, nevertheless, we find "pathological identity associated with great bacterial diversity." In a disease of this character it is not surprising to meet with symptoms varying greatly in different patients. It must also be admitted, I believe, that infective endocarditis develops different clinical features at different phases of its evolution in the same individual. If this be so, the onset of infective endocarditis will frequently date from the first attack of "acute rheumatism," "chorea," "quinsy,"[1] or other affection, at which the earliest "heart symptoms" were detected.

The two cases above quoted go far to show that this disease has periods of pathological calm, in which the recuperative powers of a patient may to some extent repair the damage produced during the active bacterial stage. As time goes on, and the patient's strength becomes more and more undermined, the intermissions usually become shorter and less complete, whilst the active stages are prolonged in duration, although seemingly less acute pathologically. If we date the commencement of infective endocarditis from the final act of this life's tragedy, as is usually done, we obtain an inaccurate, and, I consider, an altogether mistaken conception of the clinical features of this insidious malady. The assumption that the final breakdown of the circulatory apparatus in such cases is due to the sudden invasion of bacteria among the diseased tissues of a previously

[1] I have recently had a case of chorea with endocarditis at the hospital. The patient, a girl aged about 10 years, had hypertrophied tonsils. During her stay under my observation she had two attacks of tonsillitis, with high temperatures, and on each occasion an enlargement and some tenderness of the cervical gland at the right angle of the jaw. Dr. Woodhead has emphasised the important fact that the two rings of lymphoid tissue, surrounding respectively the entrances to the gullet and the windpipe, may furnish channels of infection in tuberculosis. It seems possible from the above case that in other diseases also the tonsils may serve as channels for the conveyance of the infection to the central organs.

aseptic heart, is one that cannot be substantiated either clinically, pathologically, or bacteriologically. Cases of infective endocarditis generally recover from the first attack, and, under favourable conditions, may live on for many months and occasionally for years.

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